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CHRONIC ADHESIVE PERICARDITIS

A discussion of Chronic Adhesive Pericarditis presents two classifications of clinical importance, chronic mediastinopericarditis (accretio cordis) and chronic constrictive pericarditis (concretio cordis). The two conditions may exist in combination in the same individual. A new interest in the subject has been aroused because of its more frequent diagnosis, greater perfection of surgical attack, and more favorable prognosis.

HISTORY. This subject has received recognition historically as far back as the second century. Galen in 160 A. D. recognized the fact that pericardial disease interfered with normal heart action. In 1669 Lower reported several cases of callous pericarditis; Chevers in 1843 and Wilks in 1870 gave comprehensive descriptions of the pathogenesis, clinical signs, and symptoms. In 1873 followed Kussmaul's discussion and, in 1896, that of Pick.

ETIOLOGY. The hypothesis of a previously existing infection producing inflammation and resulting in adhesions is plausible, although a large percentage of cases present no history of a definite cause. In general, etiological factors in the production of adhesive pericarditis are rheumatic fever, tuberculosis, and pneumococcal or septic infections. Of lesser importance are minor infections, cardiac infarction, syphilis, neoplasms, trauma, and scurvy. True constrictive pericarditis is not rheumatic in origin. It may occur with the polyserositis of unknown origin (Concato, 1881) or result from pneumococcal or tuberculous infection.

PATHOLOGY AND PATHOLOGICAL PHYSIOLOGY OF ADHESIVE MEDIASTINOPERICARDITIS. The adhesions formed are of importance depending upon their location, density, and extent. In accretio cordis the heart may be more or less firmly fixed to surrounding structures such as the chest wall, diaphragm, pleura, aorta, and spinal column. When dense adhesions are fixed to more or less immobile structures the work of the heart is greatly increased, resulting in cardiac hypertrophy and dilatation of the chambers involved, usually all participating in the hypertrophy. Extreme degrees of cardiac enlargement formerly attributed to chronic mediastinitis do not stand the test of careful reviewing. Cabot's series of hearts weighing over 1000 grams diagnosed as chronic mediastinitis, upon careful examination of protocols showed some accompanying valvular heart lesion (White). Coincidental enlargement of the heart frequently is due to concomitant heart disease such as pancarditis, organic valvular lesions, and hypertension. Hypertension, however, is not commonly associated with pericardial adhesions.

Hosler and Williams, reviewing 4,400 autopsy records of the University hospitals of Cleveland for the period from 1906 through 1935, reported 76 cases of extensive pericardial adhesions. In 54 cases with hypertrophy there was concomitant heart or vascular disease; in 21 cases the heart was either normal or smaller than normal; the hearts in the latter group were free from valvular disease.

In addition to mediastinopericarditis other serous membranes may show changes. Pleural effusions or fibrinous pleuritis may be present. Perihepatitis and perisplenitis may be found. Mediastinopericarditis may exist with or without polyserositis, or vice versa.

Many signs have been recorded as evidence of existing chronic mediastinopericarditis, but for the most part they are not reliable diagnostic evidence and may occur with enlarged hearts without adhesions of the pericardium. The following enumeration of these signs is sufficient: systolic retraction of the apex, systolic retraction of the left back in the region of the eleventh and twelfth ribs (Broadbent), lagging behind of the left nipple during deep inspiration (Wenckebach), fixation of the heart with change of body position, palpable diastolic shock, diastolic collapse of the neck veins (Friedreich), and paradoxical ratio in the length of time the breath can be held in inspiration and expiration (Cooper). The normal time is 40 to 70 seconds in inspiration, 20 to 25 in expiration. In mediastinal and pericardial adhesions the inspiratory holding time is nine seconds, the exhalatory 25.

Roentgenological examination may reveal irregularities in the contour of the cardiac silhouette and a decrease in amplitude of pulsations especially of the right border. Additional evidence is shown by the rise of the heart during inspiration. Bands of adhesions may be visualized. The sign of fixation of the heart upon change of position is subject to considerable error. It is of more significance when the heart is small. Retraction of the diaphragm with the heart beat has been seen.

Electrocardiographic evidence is shown by low voltage of the QRS complexes, by low amplitude or negativity of the T waves, by fixation of the electrical axis with change of body position (Carter and Dieuaide). This fixation of electrical axis with change of body position may occur, however, with a normal pericardium (Levinè).

DIAGNOSIS. The above list of symptoms and signs emphasizes the difficulty of diagnosis. Only a complete analysis of all evidence judiciously considered is of assistance in arriving at an opinion. It is a good practice always to keep adhesive pericarditis in mind, to make clinical investigations painstakingly, and to search carefully for etiological factors. Advanced heart failure with cardiac enlargement and dilatation often is most difficult to differentiate because much of the evidence available is common to both heart failure and adhesive pericarditis.

In Concretio Cordis the visceral layer chiefly is affected, the degree of thickening varying from a quarter to a half inch or more. The pericardium

may be thick and leathery, calcified, or even osseous; the two layers may be so glued together that it is impossible to separate them; and the whole heart may be encased. An association of encapsulated fluid, pus, caseous or calcareous pulp may be found. The adhesions and calcium deposits may extend into the myocardium making their separation impossible without tearing the heart muscle. In a case recently observed but not yet reported two spurs of firm calcified material 7 mm. in diameter extended into the cavity of the right ventricle for a distance of 7 mm.

Firm bands of adhesions often cause kinking and constriction of the superior vena cava, inferior vena cava, pulmonary artery, and hepatic veins. Teissier and Duvoir report serious constriction of the aorta. The location and extent of such adhesions predetermine the amount of venous engorgement in the regions so handicapped. There results either a superior or an inferior mediastinal stasis. Where the venous circulation of the liver is impaired there ensues hepatic congestion, enlargement, and finally a pseudo-cirrhosis with ascites. (Described by Pick in 1896.) While slight cirrhotic change is occasionally found, the marked changes of advanced portal cirrhosis are absent. The mechanism whereby mediastinopericarditis produces enlargement of the liver and ascites with no or only slight edema of the lower extremities is still debatable.

Due to the strangling effect of constricting adhesions the heart is normal or smaller in size. Serious constriction of the right heart chambers results more often than those of the left. One of the most serious results of constricting adhesions is the prevention of the proper diastolic filling of the heart with consequent engorgement of the large veins. Chevers pointed out that in this hypodiastolic filling of the heart the ventricles become diminished in capacity and compensate for this loss by rapid contractions. The main arteries unless diseased also adapt themselves to this diminished cardiac output. It must be remembered, however, that complete obliteration of the pericardial sac can be present without impairment of heart function.

SYMPTOMS AND SIGNS. Subjectively, precordial distress or a sense of oppression and constriction may be complained of, sometimes simulating mild angina pectoris. Palpitation and shortness of breath upon exertion should be mentioned.

Where the superior vena cava is constricted there results engorgement of the veins of the neck, head, and upper extremities with variable degrees of cyanosis. When the circulation of the liver and inferior vena cava is impaired enlargement of the liver, ascites, and lower extremity edema are found. The ascites, however, usually exists alone or is out of proportion to edema of the lower extremities.

Elevation of venous pressure, which may exceed 25 cm. of water, may exist in the presence of relative subjective well being. Systolic blood and pulse pressures are characteristically low or within normal limits. The pulse rate may be normal or rapid. The rhythm is usually regular, but there may be auricular fibrillation or extrasystoles. Pulsus paradoxus may be noted. Auscultation often reveals the presence of organic murmurs or those simulating mitral or aortic valvular disease when such valves are normal. The heart sounds, when no murmur exists, are distant or normal.

The small silent heart of true constrictive pericarditis stands out in contrast to the enlarged heart embarrassed by the anchoring adhesions of mediastinopericarditis, or that due to concomitant heart disease. Calcification of the pericardium, although rare, is pathognomonic. Schwartz in 1910 reported the first instance of calcification. Roentgen kymographic evidence presents no lateral thrust or excursion of the ventricles and is a valuable aid in demonstrating the degree of compression.

Symptoms and signs of special importance are high venous pressure, especially when the patient is able to be up and about, low arterial pressure with small pulse pressure, marked engorgement of the systemic veins, enlarged liver, a normal or a small silent heart, recurrent ascites as an outstanding feature without demonstrable cause and out of proportion to the edema of the lower extremities, engorged systemic veins predominating over manifestations of pulmonary stasis, roentgenological evidence of calcified pericardium, kymographic and electrocardiographic evidence.

COURSE AND PROGNOSIS. The course and prognosis in the cases with circulatory embarrassment are those of systemic venous congestion, enlarged liver, ascites, and cardiac failure. Patients may survive for years undergoing frequent paracentesis abdominalis and leading a life of invalidism. About middle age they usually succumb either from the effects of the pericarditis itself or from ensuing complications.

TREATMENT. Medical treatment limits itself to the complications, chief of which is circulatory failure. Here the usual measures apply. In chronic constrictive pericarditis digitalis is ineffective in controlling the venous congestion. It may be of assistance in controlling auricular fibrillation. The use of digitalis for slowing a pulse which, by its rapidity, is trying to compensate for a hypodiastolic filling of the heart may be harmful. A high protein diet may help in cases showing a low serum protein.

Different operative procedures have been devised for the two conditions. Brauer in 1902 demonstrated two cases of chronic adhesive mediastinopericarditis in which several ribs and a portion of the sternum over the precordium were removed. The heart thereby pulls on soft instead of unyielding structures. He called this procedure Cardiolysis (Thoracolysis, White). Brauer stated at that time that "the most important indication for cardiolysis is afforded by those forms of adhesive pericarditis which cause a systolic retraction of a broad area of the thorax. So long as vigorous movements of this sort are demonstrable a good result may be expected." Summers performed the first operation of this type in America.

Delorme in 1898 suggested the advisability of an operative procedure for chronic constrictive pericarditis, which consists of decortication of the heart. The heart is freed from its constricting bed by actual dissection and removal of the thickened or calcified pericardium. It is necessary to free great vessels entering the heart by releasing constricting bands. Unless adequate diastolic filling follows, the operation does not give satisfactory results. Rehn was the first surgeon to report on Delorme's suggested procedure. Sauerbruch in 1913 reported a successful case. Since then the literature presents an increasing number of cases with favorable outcome. The most astute surgical skill is necessary to carry out successfully the Delorme procedure. This is especially true when the myocardium has become softened and rendered friable by disease.

The most favorable cases for operation are those occurring in young individuals presenting the clinical triad of high venous blood pressure, ascites, and a small quiet heart. Cases in which there is an absence of concomitant heart and vascular disease also give more favorable results. Active tuberculous pericarditis is a contraindication to surgical interference. Operative procedures have produced dramatic results in properly selected cases. With improvement in diagnosis and surgical technique the field for surgery should be greatly increased.

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